located primarily in the RV outflow tract and anterior RV. But, SEA for LVARVOT were located mostly in two areas along the lateral RV base. Although locations differed, the PGs measured at the SEA were of low magnitude and were not significantly different between configurations (LVARVOT 7.1 ± 4.2 V/cm, LVASVC 7.4 ± 3.0 V/cm).

The locations of SEA leading to VF for sub-threshold DF shocks depend upon the DF electrode configuration. Regardless of the configuration, SEA are located where the PG is low and of a similar magnitude. These results support the upper limit of vulnerability hypothesis for DF.

1046-131 Where is the Best Alternate Implantation Site of the "Hot Can" Electrode?

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The "hot can" (RV to prepectoral ICD) lead system has been shown to defibrillate efficiently. A "can" electrode is implanted in the left pectoral site in most patients. However, in some patients it is impossible or inconvenient to implant at such a site. Therefore, the *purpose* of this study was to evaluate relative efficacy of alternative sites for the "hot can" electrode. *Methods:* Defibrillation thresholds (DFTs) were evaluated with a "hot can" system in random order in 7 pigs (34 ± 3.6 kg) using five different "can" electrode implantation sites: right pectoral, left pectoral, left axillary, right upper abdomen and left upper abdomen. A right ventricular coil was the cathode for the first-phase of the biphasic waveform (135 μF, 65/65% tilt) and the DFT was determined by a "down-up down-up" protocol. *Results:* Mean stored energy (joules) at DFT is shown in the table below for each "can" electrode site.

Implantation site of a "hot can" electrode

Right pectoral	Left pectoral	Left axillary	Right abdomen	Left abdomen
20.3 ± 2.7*	15.9 ± 3.8	14.9 ± 2.5	32.0 ± 3.4**	30.0 ± 3.4**

*p < 0.01, **p < 0.0001 vs left pectoral and left subaxillary site.

Conclusions: 1) DFT is depend upon the "hot can" implantation site. 2) DFT energy was unchanged in the axillary site and 30% increased with right pectoral placement. 3) DFT's for abdominal "hot can" to RV are inferior to pectorally placement ICDs.

1047 Cardiac PET

Wednesday, March 19, 1997, 9:00 a.m.–11:00 a.m.
Anaheim Convention Center, Hall E
Presentation Hour: 10:00 a.m.–11:00 a.m.

1047-141 PET Myocardial Perfusion Imaging Significantly Reduces the Cost of Coronary Disease Management

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We hypothesized that myocardial perfusion imaging (MPI) with PET and Rb-82 results in cost savings in coronary disease (CAD) management, compared with SPECT MPI, due to improved test accuracy, despite the increased cost of PET. Accordingly, we compared procedure costs and outcomes in 303 sequential patients referred for CAD evaluation with PET MPI, and in 102 patients imaged with SPECT, matched for pretest likelihood of disease with "CADENZA" software, which incorporates type of chest pain, cardiac risk factors, comorbid conditions, and previous exercise test results. Costs were calculated using estimated charges: SPECT-$1000, PET-$1850, Angio-$5000, PTCA-$10,000, CABG-$40,000. At least three month follow up was obtained in all patients.

There were no deaths in the PET group. One myocardial infarction occurred just prior to angiography, which was recommended due to a high risk PET result.

The percentages of angiography, CABG and PTCI for SPECT vs. PET were: 32.3 vs 9.8; 7.8 vs 3.3; and 2.0 vs 1.7 respectively.

	n	Pretest Probability	Diagnostic Cost/Pt	Treatment Cost/Pt	Total Cost/Pt
SPECT	102	0.36 ± 0.14	$2,617.65	$3,333.33	$5,950.98
PET	303	0.37 ± 0.17	$2,380.07	$1,724.48	$4,104.55

Conclusion: A management strategy using PET-MI in patients with an intermediate risk of CAD results in: reduced angiography and revascularization procedures, excellent short-term patient outcomes, and a 30% cost-savings when compared to conventional management with SPECT imaging.

1047-142 Simultaneous Myocardial Blood Flow Estimation Using 13N-NH3-PET and the Argon Inert Gas Technique in Humans

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Purpose of the study was to validate the quantification of myocardial blood flow (MBF) with 13N-NH3 and positron emission tomography (PET) with the argon (Ar) inert gas methode. Estimation of MBF using NH3-PET and the application of a two compartmental model was validated in animals using 15H-H2O PET and the microspheres technique. In humans, calculated values using this approach seemed to be reasonable but were not validated.

We investigated 12 patients (7 male, 5 female) who had undergone coronary angiography (relevant coronary stenoses were excluded) and were suspicious for small vessel disease. MBF was simultaneous assessed after injection of 740 ± 110 MBq 13N-NH3 using dynamic PET (ECAT 931 or ECAT Exact HR+) and by the argon (Ar) inert gas method. Measurements were performed at rest and during vasodilatation (0.80 mg/kg/4 mins diypridamole or 0.14 mg/kg/min adenosine for 15 mins).

In 21 investigations (9 stress, 12 rest) estimation of MBF were successful for both modalities: flow values were 0.98 ± 0.16 ml/g/min and 0.90 ± 0.24 ml/g/min at rest and 2.23 ± 0.97 ml/g/min and 2.32 ± 0.73 ml/g/min during vasodilatation for Ar and PET, respectively. These values are statistically not different (paired t test: $p > 0.05$). Correlation of both methods was $r = 0.89$. High MBF during rest was due to increased breathing work and excitation (suspected from a rate pressure product of 9.428 ± 2.149). Coronary flow reserve was 2.3 ± 0.9 and 2.9 ± 1.4 for Ar and PET, respectively.

For the first time in vivo MBF estimation using NH3-PET and a two compartment model was validated with the argon inert gas method in humans.

1047-143 Predictive Value of PET Blood Flow/Metabolism Mismatch in Patients with Type II Diabetes Mellitus

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The accuracy of PET blood flow/glucose metabolism imaging for predicting the postrevascularization improvement in regional LV function in CAD patients ranges from 72–95% in unselected patients, yet remains undefined in those with diabetes mellitus (DM). Therefore, we studied 20 consecutive patients (mean age 64 + 11 years; 8 with DM = group I; 12 without DM = group II) with PET ^{13}N-ammonia and ^{18}FDG 2 ± 2 weeks prior to revascularization. The LVEF did not differ between both groups (35 ± 8% vs. 29 ± 7%). Flow/metabolism mismatches in the LAD, LCX and RCA territories were identified by polar map analysis. Regional and global LV function was determined by 2D echocardiography 14 ± 14 days prior to and 150 ± 145 days after revascularization. Changes in regional wall motion by ≥ 1 grade on a scale of 4 (0 = akinesis and 3 = normal) were considered significant. Fifty-two territories were analyzed; 23 were classified as mismatch, 13 as match and 18 as normal.

Presence of mismatch and improvement in regional LV function

	Sens	Spec	PPV	NPV	Acc
Group I	92%	63%	80%	83%	81%
Group II	38%	87%	50%	80%	74%

LVEF increased from 29 ± 6% to 40 ± 7% ($p < 0.01$) in patients with ≥ 2 mismatch territories but not in patients with only one mismatch territory (34 ± 11% vs. 36 ± 10%, $p = 0.5$). Thus, if ≥ 2 mismatch territories were considered a prerequisite for an improvement in LVEF by at least 5%, the positive and negative predictive values were 80% and 100% in group I and 67% and 72% in group II. In conclusion, the predictive accuracy of PET blood flow/metabolism imaging is maintained in patients with DM. Moreover, regulation of glucose utilization in reversibly injured myocardium appears to be preserved by local factors despite insulin resistance.

1047-144 The Measurement of Myocardial Blood Flow With Positron Emission Tomography is Highly Reproducible

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Positron emission tomography (PET) is considered the gold standard for the non-invasive quantification of myocardial blood flow (MBF) in man. However, since little is known about the reproducibility of MBF measurements with PET, we aimed to study baseline and hyperemic MBF twice in the same patients on two different occasions. *Methods:* Thirty patients with angiographically

normal coronary arteries were studied; 14 pts with cardiac syndrome X (SX, 9 F, age 52 ± 7 years) and 16 pts with hypertrophic cardiomyopathy (HCM, 4 F, age 41 ± 12 years). The clinical status of all pts was unchanged between the two PET studies. The MBF tracer was O15-water for SX and N13-ammonia for HCM. MBF (ml/min/g) was measured at baseline (bas) and after dipyridamole (dip; 0.56 mg/kg in 4 min) and the CVR computed as dipMBF/basMBF (absolute units). The coefficient of variation (CV%) of MBF was calculated as SD/mean of regional basMBF and dipMBF. *Results:* Mean time between studies was 218 days, range 10–682, for SX and 841 days, range 38–1765, for HCM. No differences were found between the first and second study in SX and HCM for measured and derived MBF parameters (ANOVA, p = NS; table).

	SX 1st study	SX 2nd study	HCM 1st study	HCM 2nd study
basMBF	1.16 ± 0.41	1.20 ± 0.26	0.84 ± 0.27	0.76 ± 0.31
dipMBF	3.11 ± 1.08	3.65 ± 1.13	1.35 ± 0.47	1.40 ± 0.69
CVR	2.71 ± 0.63	3.08 ± 0.80	1.68 ± 0.54	1.94 ± 0.69
basCV	18 ± 9	16 ± 8	13 ± 9	14 ± 9
dipCV	27 ± 13	21 ± 7	17 ± 12	16 ± 9

Conclusions: The results of the present study show that PET offers reproducible non-invasive measurements of MBF in humans.

1047-145 Effects of Left Bundle Branch Block on Cardiac PET Imaging

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To evaluate the effects of left bundle branch block (LBBB) on perfusion and metabolic cardiac PET imaging, 20 patients (18 M, 2 F, aged 51–74 yrs, mean 64) affected by permanent, complete LBBB were enrolled and submitted to: 1) glucose load F18-fluorodeoxiglucose PET (FDG-PET); 2) rest N13-ammonia PET (NH3-PET); 3) coronary angiography; 4) echocardiography. Eleven pts presented a history of a previous myocardial infarction, anterior in localization in 4 and infero-posterior in the other 7 at the echocardiography. The other 9 pts presented a left ventricular dilatation, without signs of previous necrosis. At the coronary angiography 12 pts presented a significant stenosis of LAD, 8 of these with involvement of other coronaries, while 2 pts presented only stenoses in RCA and LCx and in the other 6 pts no significant coronary stenosis was found. At the FDG-PET all the LBBB pts presented a severe uptake defect (< 50% of the maximum) in the septum, extending to nearby regions of the anterior and inferior left ventricular wall, overall involving from 40 to 60% of the ventricle wall. In the corresponding area the perfusion, evaluated by NH3-PET, was preserved. At the echocardiography the wall motion segmental analysis revealed no significant correlation between the dysfunctional area and the site of the FDG uptake defect.

In conclusion our study could suggest that: 1) in LBBB a change in the metabolic activity of the septum without damage in perfusion was present; 2) this was evident also in the pts without LAD stenosis; 3) in LBBB pts a viability study using FDG-PET may overestimate the extension of the necrosis, to avoid this misinterpretation it appears advisable to perform a perfusion PET study using N13-NH3 as well.

1047-146 Delayed FDG Imaging Improves Myocardial/Blood Pool Contrast

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In some pts with CAD, imaging of the myocardium with F-18 deoxyglucose (FDG) at 30–60 min post injection by PET produces images with low contrast between myocardium and blood pool. We hypothesized that imaging at a later time might improve the quality of FDG images both by increasing myocardial uptake and clearance of FDG from the blood. We tested this hypothesis in 7 pts with CAD: 6 men, 1 woman, age 66 ± 11; 3 diabetics. PET images were acquired both at 30–60 min post injection of 57–10 mCi of FDG (1 hr after glucose load) and at 135–165 min. For each pt, the entire LV was divided into 41 approximately equal segments. Both absolute (nCi/cc/mCi) and normalized (to peak thallium activity on stress) FDG uptake were analyzed. All data were decay corrected to time of injection. Normalized early and late FDG values were highly correlated (slope = 1.26, intercept = −0.25, r = 0.92, SEE = 0.05), implying early and late images provide similar clinical information. Of 215 segments, 208 (97%) were concordant for viability between early and late imaging. Myocardial FDG uptake increased on late imaging (77 ± 27 early vs 116 ± 57 late, p < 0.05). In all pts, late imaging was associated with decreased blood pool activity (34 ± 6.6 early vs 18 ± 7.1 late, p < 0.01), resulting in improved late myocardium/blood pool contrast ratio (2.3 early vs 7.1 late, p < 0.01). This improvement in myocardial contrast was similar for diabetics (from 2.1 to 6.5) and non diabetics (from 2.5 to 7.6), and was consistent with marked visual improvement. These data suggest that despite a slight decrease in myocardial counts due to decay, the increased myocardial FDG uptake combined with decreased blood pool activity improves late image quality. The good early-late FDG correlation suggests that the improvement does not alter the FDG distribution in a clinically significant way, but this needs to be verified in a larger number of pts.

1047-147 Predictive Value of FDG Imaging in Patients With Chronic Ischaemic Left Ventricular Dysfunction Enrolled in a Prospective European Multicentre Viability Study

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Aim of this study was to ascertain the value of quantitative PET with ^{18}F-fluorodeoxyglucose (FDG) to identify hibernating myocardium. A total of 502 patients (pts) with at least one dysfunctional (D) segment (S) subtended by a stenotic coronary artery amenable to revascularization (R) were enrolled over 2 years. Left ventricular (LV) function was assessed before and 4–6 months after R by radionuclide ventriculography or echocardiography. To maximize myocardial FDG uptake and overcome insulin resistance, all studies were performed during euglycemic hyperinsulinemic clamp (EHC). A total of 254 pts have undergone R, by angioplasty (14%) or bypass. Complete follow up data on 121 pts (age 59 ± 9) who had R are available: LV ejection fraction (EF) was 37 ± 13% before and 40 ± 15% after R (p < 0.01); the regional wall motion score (1 = normal; 2 = hypokinetic; 3 = akinetic; 4 = dyskinetic) was 2.0 ± 0.6 before and 1.7 ± 0.6 after R (p < 0.001). A total of 326 S were normal (N) and 382 D. After R, 204 (53%) D-S improved (IMP), 149 (39%) were unchanged (UNC) and 29 (8%) worsened. The Metabolic Rate of Glucose (MRG; μmol/min/g; lump constant = 1) was 0.42 ± 0.18 in N-S, 0.38 ± 0.17 in IMP-S (p < 0.01 vs N) and 0.31 ± 0.17 in UNC-S (p < 0.01 vs N and IMP). *In conclusion,* these interim results indicate that FDG-PET during EHC can provide the quantitative assessment of myocardial viability in the absence of perfusion measurement.

1047-148 Myocardial Blood Flow Changes During Low *Dose Dobutamine Infusion: Relation to F-18* Deoxyglucose Uptake

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In asynergic myocardial regions rendered dysfunctional by chronic hypoperfusion, F-18 deoxyglucose (FDG) is useful in differentiating viable from scarred myocardium. In this study, we determined whether regional myocardial blood flow (MBF) response during low dose dobutamine (LDD) infusion also differentiates hypoperfused but viable from scarred myocardium when compared to FDG uptake. To this end, 11 pts (mean age 65 ± 9 years) with chronic CAD and LV dysfunction (mean LVEF = 32 ± 6%) underwent positron emission tomography (PET) with N-13 ammonia (NH_3) and FDG. Absolute MBF was computed at rest and during LDD (5 μg/kg/min) infusion, using a two compartment NH_3 model. Four to five NH_3 and FDG PET slices (8 regions per slice) were matched and analyzed for each patient. Rest and LDD N-13 ammonia data were then analyzed according to the magnitude of FDG uptake.

FDG (% uptake)		N-13 Ammonia (ml/min/g)			
		Rest		Low-dose Dobutamine	
Normal	(≥ 80%)	0.64 ± 0.18		0.98 ± 0.32	
Moderate ↓	(50–79%)	0.47 ± 0.22	] p = NS	0.82 ± 0.38	] p < 0.001
Severe ↓	(< 50%)	0.37 ± 0.21		0.57 ± 0.25	

At rest, MBF was similar in regions with moderately and severely reduced FDG uptake. During LDD infusion, although mean MBF increased in all three categories, the extent of MBF increase was significantly greater in regions with moderately than in regions with severely reduced FDG uptake (0.35 ± 0.26 vs 0.20 ± 0.30 ml/min/g, p < 0.05). Furthermore, 85% of regions with moderately reduced FDG uptake showed mismatch pattern (FDG: MBF ≥ 110). *These data suggest that an assessment of MBF response during LDD* infusion may differentiate hypoperfused but viable from nonviable myocardial regions.